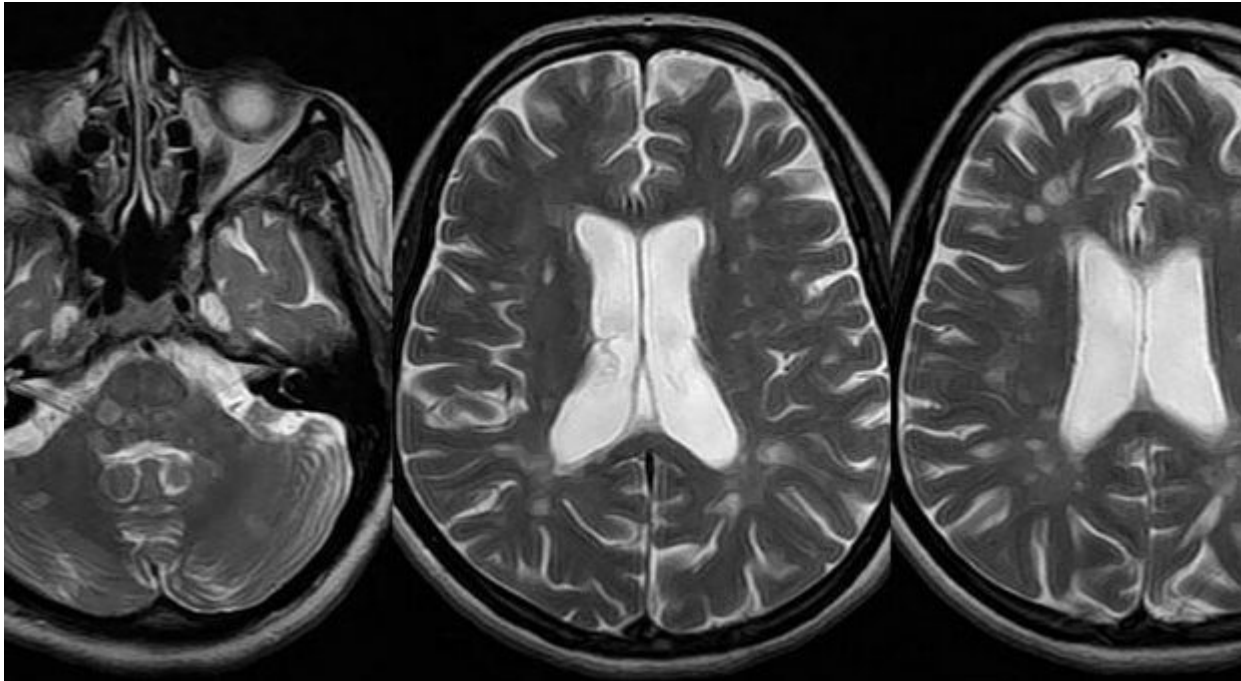


# Progressive MS: Looking for Answers Now



**People who live with progressive MS have many questions, but one I hear often is, “When will there be treatment options for me?” Based on what I saw and heard at last week’s AAN, I’m pleased to report that researchers from around the world are making important progress toward treatments and therapies for people living with progressive MS.**

Several groups presented results or updates from large, ongoing studies involving people living with primary-progressive MS: one, [a study of oral laquinimod](#), an experimental immunomodulator, in 375 people with primary-progressive MS which recently began recruitment ; [second, a clinical trial of oral ibudilast](#), an anti-inflammatory enzyme used in Japan, recruiting 250 people with primary- or secondary-progressive MS; and third, [a study of ocrelizumab](#) – an antibody cousin of rituximab delivered by infusion – in 740 people with primary-progressive MS that has completed enrollment. No results are available yet, but some should be next year, and it’s encouraging to see that these trials are getting under way. I hope the findings provide us with new treatment approaches for people with progressive MS. (Abstracts # P7.210, P7.017)

One aspect of research into treatments for progressive MS may not seem very intuitive: Sometimes scientists need to test treatments in people with very early signs of the disease, such as optic neuritis, an inflammation of the optic (eye) nerve that is often the first symptom of MS, much before progression is even evident. They do this because the nerve damage – and if successful, the repair or protection from damage – is more easily observed in this single location.

That brings us to exciting results from a trial led by Dr. Raju Kapoor (University College London), which recruited 86 people with optic neuritis. They were randomly assigned to receive either phenytoin – an FDA approved oral therapy used to treat epilepsy – or a placebo for 3 months to assess whether the phenytoin could help to protect the retinal nerve fiber layer at the back of the eye from damage. Of those completing the study, on average people who received phenytoin had 30% less damage to the nerve fiber layer compared to those who received placebo. The results raise the possibility of “repurposing” a therapy already on the market with a long track record of use. We need to confirm these results in a larger study to really understand if phenytoin can truly protect the nervous system from damage that leads to MS progression. (Abstract # PL2.005)

In another study, 154 people with primary- or secondary-progressive MS were given experimental MD1003 (concentrated biotin, a B vitamin), or an inactive placebo, for 48 weeks. The results showed that 12.6% of those given MD1003 showed improvement in disability, using the EDSS scale that measures disability progression or improvement in a timed walk, versus none of those on placebo, and there were no serious safety issues reported. More research is needed to figure out who might benefit from this approach and why only 12% responded. The manufacturer, MedDay Pharma, says that another trial is underway in people with MS and results are expected later this year. (Abstract #PL2.002)

Finally, I was impressed with a study from Dr. Mika Komori and a team at the National Institutes of Health that looked “behind the scenes” to try and better understand why immune-modulating treatments have not succeeded in progressive forms of MS as they have in relapsing MS. The team examined spinal fluid samples from 386 people with all types of MS as well as people without MS, to determine the exact numbers and characteristics of various immune cells. What they observed is that attacking immune cells in people with progressive MS were more likely to be holed up in the brain and spinal cord, whereas the cells in people with relapsing forms were mobile and circulating. What does this mean? It may be that, for treatments to succeed at modulating inflammation and/or nerve damage in progressive MS, the therapies will have to be able to track the bad cells within the central nervous system. (Abstract #S12.001)

Everyone with MS lives with the uncertainty of whether it will progress and whether they will lose the ability to do the things that matter most to them. I’m encouraged by the research I saw last week and strongly believe this kind of research will drive us to find ways to stop progression and restore or repair lost function.